The hard palate presented a similar appearance, but the soft palate and pharynx showed no abnormal pigmentation. There was pigmentation of the inner side of the right cheek, extending as far back as the teeth on that side. There was no pigmentation of the laryngeal mucous membrane, matrix of nails, scrotum, penis, anal folds, or axillæ.

Thyroid gland was not enlarged. Radial pulse was almost imperceptible; expansion very slight; vessel wall soft.

There was nothing of note in the examination of the lungs, heart, liver, or spleen. The abdomen was retracted; pulsation of the abdominal aorta was distinctly visible and palpable. Examination of the urine was negative.

The patient died two days after admission. The autopsy showed isolated primary tuberculosis of both adrenal glands, with incipient tuberculous meningitis.

The chief point of interest in this case was the fact that there was no tendency on the part of the discrete pigmented areas to run together and become diffuse. The patches of pigmentation remained isolated throughout up to the time of death. Although the onset of pigmentation in Addison's disease is occasionally in the form of discrete patches, yet in nearly all these cases the pigmentation becomes diffuse before the fatal termination of the case.

Trebitsch states that these cases of Addison's disease might be mistaken for various affections in which pigmentation of the skin occurs. Among these he mentions the idiopathic multiple sarcomata of Kaposi, xeroderma pigmentosum, pigmented lesions following syphilis, and lentigines.

Changes in Metabolism and the White Blood-Corpuscles in Blood Dissolution.—KUHNAN (*Deutsch Archiv f. klin. Medicin*, 1897, Band lviii. 4 u. 5 Heft) studied the changes in metabolism and white corpuscles in diseases associated with dissolution of the red cells, and arrives at the following conclusions:

1. In blood dissolution there are marked leucocytosis and alterations in metabolism. The metabolic changes consist in:
    - (*a*) An increase in the uric acid and xanthin bases elimination.
    - (*b*) An increase in the excretion of phosphoric acid at first, followed by marked diminution later.
    - (*c*) An increase in the elimination of the chlorides.
    - (*d*) An establishment of a vicarious relationship between the chloride and phosphoric acid elimination, the retention of one being associated with an increased excretion of the other.
2. The increase in the alloxuric bodies (uric acid and xanthin bases) is dependent on an increased destruction of leucocytes.
3. The mother substance from which the alloxuric bodies are formed is contained chiefly in the plasma and serum of the blood, and to a much less extent in the red cells.
4. The amount of the uric acid forming substance is dependent on the richness of the blood in leucocytes.
5. The leucocytosis in blood dissolution is the result of the combined action of the following components:

(*a*) The direct leucotactic action of the blood poison.

(*b*) The action of the chemical substances (particularly the nuclein substance) resulting from the destruction of the corpuscular elements of the blood.

6. The leucocytosis resulting from the injection of corpuscular elements into the blood stream is a pure phagocytosis, and does not lead to any marked destruction of the leucocytes.

7. The increased leucocyte destruction in infectious diseases is due to the presence in the blood of a substance which destroys the vitality of the leucocyte.

SURGERY.

UNDER THE CHARGE OF

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Hypertrophy of the Gums.—HEATH (*British Medical Journal*, May 1, 1897) reports a remarkable and somewhat uncommon case of hypertrophy of the gums in the person of a young man, aged twenty-six years. Hypertrophy of the upper lip and a fulness of the cheeks were the most prominent features when the mouth was closed, but when the mouth was opened it reminded one of the mouth of a hippopotamus or rhinoceros on a small scale.

The history shows that the hypertrophy was first noted about four years previously, when there was a comparatively slight hypertrophy of the gums and no displacement of the teeth. This hypertrophy was removed at that time by operation. Three months later the patient noticed the growth again, and it has steadily increased during the last three years.

The external deformity was well marked on admission, and the gums were seen to be enormously hypertrophied when the mouth was open, and most of the teeth were loosened and displaced. The palate looked at first like a cleft palate, but this was due to the hypertrophied gum on each side covering the palate nearly to the median line, where a small interval was left.

On account of the free bleeding experienced in a former case, the patient was placed on a table with the head falling off. The teeth were then rapidly extracted—all the teeth of the upper jaw except two canines, which were firm—and the hypertrophied gums cut away with scissors down to the alveolus, the edge of which was removed by the bone-forceps. The bleeding was checked by the Paquelin and by plugging.